



A CASE CONTROL STUDY ON THE PATTERNS BLOOD PRESSURES AND AEROBIC CAPACITY AMONG YOUNG ADULT SMOKERS.

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ABSTRACT

Introduction: - India is home to about 12% of the world's smokers. More than 1 million people die every year due to tobacco related illnesses. Smokers have tendencies to be less physically active than non-smokers. We hypothesises that there was a significant relation between smoking and decrease in cardiovascular fitness. The purpose of this study was to examine the chronic effects of smoking on cardiovascular fitness and Blood pressure in young and adult smokers. **Methods:** This was case control study conducted in a span of 3 months in local General Medicine OPDs. 80 Subjects chosen for the study (40smokers & 40 non-smokers) involving both the genders, aged 18 years to 25 years and all classes of socio economic strata. 80 male participant were recruited & divided into two group 40 smoker (A) , 40 non-smoker (B). BP & Heart rate (HR) were non-invasively measured in young non-smoking and subjects at rest, during the accomplishment of a sub maximal exercise test and recovery period. Smoking status of smokers was analyzed by three parameters, i.e., years of smoking, number of cigarettes smoke per day, and pack year. **Result:** There was no significant difference between both groups regarding to age, weight and height. The mean duration of smoking among smoker group was 4.84 ± 2.57 years. No. Of Cigarettes were 4.62 ± 2.35 SBP, DBP & PP were pre-hypertensive, 40% were hypertensive. There was Significant difference w.r.t Resting Heart Rate, HR after Exercise, Recovery HR & Exercise duration among smoker and non-smoker group. **Conclusion:** Smoking was found to affect young smokers' HR, increasing HR at rest, slowing HR increase during exercise, and impairing their ability to reach the age predicted HR max. In addition, smoking was associated with an attenuated HR decline during recovery. Smokers had a higher resting HR and showed a higher HR response during sub maximal exercise compared to Non smokers.

KEYWORDS :Smokers, aerobic capacity, Blood pressure, Heart rate.

INTRODUCTION

Smoking in India has been known since at least 2000 BC when cannabis was smoked and is first mentioned in the Atharvaveda (compiled c. 1200 BC- c. 1000 BC). Tobacco was introduced to India in the 17th century. It later merged with existing practices of smoking (mostly of cannabis). Smoking in public places was prohibited nationwide from 2 October 2008. There are approximately 120 million smokers in India. According to the World Health Organization (WHO), India is home to 12% of the world's smokers. More than 1 million people die every year due to tobacco related illnesses.¹ As of 2015, the number of men smoking tobacco in India rose to 108 million, an increase of 36%, between 1998 and 2015²

According to a 2002 WHO estimate, 70% of adult males in India smoke. Among adult females, the figure is much lower at between 13- 15%.¹ About 90% of children under the age of 16 years (10th class) have used some form of tobacco in the past, and 70 % are still using tobacco products.¹ Smokeless tobacco is more prevalent than cigarettes or bidis in India.¹

According to the study, "A Nationally Representative Case-Control Study of Smoking and Death in India", tobacco will be responsible for 1 in 5 of all male deaths and 1 in 20 of all female deaths in the country by 2010. This means approximately 1 million Indians would die annually from smoking by 2010.³ According to the Indian Heart Association (IHA), India accounts for 83% of the world's heart disease burden, despite having less than 20% of the world's population. The IHA has identified reduction in smoking as a significant target of cardiovascular health prevention efforts.⁴

A survey conducted by the International Institute of Population Science and the Ministry of Health and Family Welfare, reveals that 56.6% of people in Kolkata smoke, the highest rate in the country. 82% of men and 23.5% of women smoke in Kolkata⁵ The highest number of bidis smokers are in Uttarakhand.

It is acceptable worldwide that cigarette smoking is the leading cause of preventable death all over the world. Recent statistical analysis tales that it will be very difficult to reduce tobacco-related deaths in next 30- 50 years unless tobacco users are encouraged to quit. It. 6 The hazardous effects of cigarette smoking on general health are well documented, and there is evidence that cigarette smokers die 10 years younger than non-smokers. 7, 8 Cigarette smoking is a powerful addiction, so experimentation and uptake of smoking which typically occur in late child hood or adolescence. 9 Cardiovascular diseases are the leading cause of death is accepted globally.

10 Cardiovascular diseases contribute to one-third of death globally and are a leading cause of disease burden in developed and developing countries¹¹ cardiovascular diseases are eminently preventable diseases. Therefore, to reduce the burden of cardiovascular disease, the strategies should target the life style-related risk factors such as unhealthy diet; tobacco smoking, physical inactivity as well as morbid conditions arise due to these factors such as hyperlipidemia, hyperglycaemia, or hypertension. Hypertension is a preventable risk factor for cardiovascular disease which is very common health problem worldwide due to the contribution of risk factors such as cigarette smoking and physical inactivity. 12, 13 Hypertension plays a major etiologic role in developing cerebrovascular disease, ischemic heart disease, and renal failure. Proper treatment of hypertension is essential to reduce the burden of diseases. 14 It is also studied that carebrovascular disease, ischemic heart disease, renal failure, and other disease are associated not only with hypertension alone but also with suboptimal blood pressure (BP). 15 Previous data suggest that carebrovascular disease and ischemic heart disease occur in persons with suboptimal BP (systolic BP [SBP]>115mmHg). 11 Several studies have been done to establish relation between cigarette smoking and BP. Some studies reported that cigarette smoking decreases BP; some reported smoking increases BP. Young Adults and students are the future

valuable as health and wealth of the nation; they should have a healthy life. Thus, this study will help prevent the health hazardous of young adults exposed to smoking.

Cigarette smoking has long been known as a serious topic in public health. And it has been increasing in many developing countries around the world. 16 Smoking is a major risk factor for cardiovascular morbidity and mortality, and was considered to be the leading preventable cause of death in the world. 17 Tobacco has more than 4,720 chemical products, nicotine was being the responsible for addiction, increase of heart rate, blood pressure 18 and double product (DP) 19 which are alterations associated with the increase of cardiac work in smokers. 20,21 The European Society of Cardiology reported recently that smoking causes 28% of cardiovascular deaths in men aged 35 to 69 years and 13% in women of the same age. 22 Smoking either active or passive, can cause cardiovascular disease via a series of interdependent processes, such as enhanced oxidative stress, hemodynamic and autonomic alterations, endothelial dysfunction, thrombosis, inflammation, hyperlipidaemia, or other effects. 22. Exercise tolerance, expressed as the peak workload achieved during a maximal treadmill exercise test, is a leading indicator of circulatory system capacity as it is strongly related with maximum O₂ uptake 23, which is an excellent non-invasive measure of maximum cardiac output and exercise capacity^{24,25}.

It was well documented that smokers have tendencies to be less physically active than non-smokers 26. So we hypothesises that there was a significant relation between smoking and decrease in cardiovascular fitness the purpose of this study was to examine the chronic effects of smoking on cardiovascular fitness and Blood pressure in young and adult smokers 27.

METHODOLOGY

This was a case control study conducted in a span of 3 months in local General Medicine ODPs. It involved Prior Consent from all the Subjects & the Hospital Authorities /Medical Superintendent of the Health Care Units. 80 Subjects chosen for the study involving both the genders, aged 18 years to 25 years and all classes of socio economic strata. Subjects were explained the protocol and output of the study. They were told that confidentiality will be maintained.

Selection of participants was done by history taking and general health check up. Smoker group included subjects who were current smoker with minimum 6 months history of cigarette /bidi/hukka smoking.

The exclusion criteria included patients who were having Diabetes mellitus or impaired fasting glucose or impaired glucose tolerance, Any past History of CAD or CVA, any Renal diseases and any other endocrinal diseases like Hypothyroidism or Hyperthyroidism.

Non-smoker group included students who have never smoked cigarette or use any other tobacco products. Students with associated systemic disease, family history of hypertension, diabetes, and endocrinal diseases were excluded from this group. Occasional smokers were excluded from either of the group.

Data of duration of smoking in years and number of cigarettes smoke per day were collected from each smoker. These two data were helpful to calculate pack year by the following formula- No. of pack years= No. Of cigarettes smoked per day x No. of years smoked/20.28

For Pattern of BP:

The BP of each participant was recorded in auscultatory

method by the instrument. (also known as Riva Rocci Korotkoffs or Manual for BP measurements). Participants were instructed to take normal diet along with to avoid any medication or drug previous night of BP recording. They were also instructed to take minimum 6-8 h of sleep of previous night of BP recording. BP was recorded in the morning between 7 and 8am. Before recording, they were given 5-10min of rest. Then, BP was recorded in sitting position, the right arm of the participant by auscultatory method. Three recordings were taken from each participant, and the average of three was taken both for SBP and diastolic BP (DBP). Pulse pressure (PP) was calculated from SBP and DBP (PP=SBP-DBP). BP of both smoker and non-smoker was classified as normal, pre-hypertension, according to the Joint National Committee⁷.

Randomization was done. Essential Hypertension of any grade as defined by the Seventh report of the Joint National Committee 7 (JNC 7) guidelines & accordingly to JNC 7 criteria, stage 1 and stage 2 were considered for this study. All patients were subjected to detail history after taking written and informed consent and detail systemic examination. They were subjected to detailed history and physical examination (including vitals, weight, height, and body mass index [BMI]), with special emphasis on the examination of cardiovascular system.

Other necessary biochemical investigations were also done For Aerobic Capacity:

This study was done in local Medicine lab, 80 male participant were recruited & divided into two group 40 smoker (A), 40 non-smoker (B). Heart rates (HR) were non-invasively measured in young non-smoking and smoking subjects at rest, during the accomplishment of a sub maximal exercise test and recovery period.

Measurements

- Heart rate measured manually
- Exercise test unite (treadmill and ECG monitor)

All subjects were underwent a sub maximal Bruce treadmill test and their HR was recorded during, at peak, and after termination of exercise.

Exercise test

1. All participants were exercised with the standard Bruce treadmill test. The sub maximal exercise test was used as an indirect measure of participants' exercise capacity.
2. Age-predicted target HRs were used as predetermined endpoints.
3. Testing was terminated at sub maximal effort or when symptoms such as intense exhaustion, fatigue, dyspnoea, or intense leg pain occurred.
4. All subjects were placed sitting immediately after termination of the exercise test for a 5-min recovery period.

Data was filled in Microsoft Excel & analysed using a computer software Epi Info version 6.2 (Atlant, Georgia, USA) & SPSS. Chi-square test was used to analyze nonparametric or categorical data. For analysis of ordinal scale data, Student's t-test was used. Karl-Pearson correlation coefficient was calculated to observe correlation between variables.

P<0.05 was taken as significant and <0.01 as highly significant. P value of 0.05 and less was considered as statistically significant.

Smoking status of smokers was analyzed by three parameters, i.e., years of smoking, number of cigarettes smoke per day, and pack year, and the data were expressed in minimum, maximum, and mean \pm standard deviation form. BP of smoker and non-smoker was compared by unpaired t-

test. BP of smokers was analyzed and classified as normal, pre- hypertension, and hypertension. Smokers were distributed in groups, i.e., normal, pre-hypertension, and hypertension, and data were expressed in number and percentage form. P<0.05 was considered to be significant. Generation of tables and graphs was done by Microsoft excel and word.

RESULTS

This study was done to see the effect of smoking in local young adult's w.r.t the patterns of BP & their Aerobic capacity. All the participants recruited in this study were male and between the age of 18-25 years. A total number of participants included in this study were 80, which included 40 smokers and 40 non-smokers.

Table1. shows the smoking position of smokers

Study Variables	Maximum	Minimum	Mean
Duration of Smoking	9 years	6 month(0.5 years)	4.84 ±2.57 years
No. of Cigarettes per day	14	2	4.62± 2.35
Pack year	3.4	0.2	0.92 ±0.46

In group A (smoker) was composed of 40 subjects their mean age, weight and height were 21.2±2.31 years, 72.8±5.2 Kgs, 171.9±4.2 cm respectively.

In group B(non-smoker) was composed of 40 subjects their mean ages, weight and height were 21.4±2.2 years, 73.6±5.4Kgs, 172.4 ± 5.1cm respectively.

There was no significant difference between both groups regarding to age, weight and height, with P value more than 0.05. (Table2)

Table2. Difference between both groups regarding to age, weight and height, with P value

BP Levels	Smokers(A)	Non Smokers (B)	P value
SBP	136±8.53 mmHg	118±7.92 mmHg	Significant (0.0005)
DBP	87±5.6 mmHg	80±7.5 mmHg	Significant (0.01)
PP	48.62±7.2 mmHg	37.7±6.9 mmHg	Significant (0.0001)

	Age in years	Weight	Height
Group A	21.2±2.31	72.8±5.2	171.9±4.2 cm
Group B	21.4±2.20	73.6±5.4	172.4±5.1
P value	Not Significant	Not Significant	Not Significant

Table 3 showing The comparison of BP between smoker (A) and non-smoker (B)- Mean SBP of smoker was 136±8.53 mmHg and non-smoker was 118±7.92mmHg. This difference in BP was significant at P less than 0.05Mean DBP of smoker was 87±5.6 mmHg and non-smoker was 80 ± 7.5 mmHg. This difference in BP was significant at P value less than 0.05. Mean PP of smoker was 48.62±7.2mmHg and non-smoker was 37.7±6.9mmHg. This difference in BP was significant at P less than 0.05.

Table3. Comparison of BP between smoker (A) and non-smoker (B)

(SBP-Systolic Blood Pressure, DBP- Diastolic Blood Pressure, PP- Pulse Pressure)

The distribution of smokers into different categories on the basis of severity of BP was out of 40 smokers, 5 had normal BP

which accounts only 12.5% smokers, 19 were pre hypertensive which accounts 47.5% of smokers, and 16 were hypertensive which accounts 40% of smokers.

About the aerobic capacity, Table4- b Showing the significant change between both groups regarding to resting heart rate in groups A and B (77.7±4.16 beats per minute(bpm) versus 71.4±4.85 bpm) with P value 0.0001. Regarding to heart rate after exercise in group A and B, it was (134.4±6.5 versus 129.6±7.4) with P value; 0.006, regarding to heart rate after 5 minute recovery from exercise in group A and B, it was (84±7.7 versus 83±7.4) with P value; 0.578

Regarding to time exercise in group A and B, it was (4.8±0.8 versus 6.4±0.9) with P value 0.0001

Table-4

	HR at Rest (bpm)	HR after Exercise (bpm)	HR after 5 minutes Recovery	Exercise Duration
Group A (Smokers)	77.7±4.16	133.5±6.5	83±5.7	4.7±0.5
Group B (Non Smoker)	71.4±4.85	128.6±7.4	82±4.1	6.2±0.9
P Value and Significance	0.0001, Significant	0.006, Significant	0.578, Not Significant	0.0001, Significant

DISCUSSION

A lot of studies were done to find the effect of cigarette smoking on BP, and a lot of controversies were there. Some studies reported smoking rises BP; some reported smoking decreases BP while some reported no effect. However, this study reported that SBP, DBP, and PP of smokers were more than non smokers. Further detail analysis of BP of both smoker and non-smoker was done. There was no significant difference between both groups regarding to age, weight and height, with P value more than 0.05.(Table-2)

Mean SBP of smoker was 136 ± 8.53 mmHg and non-smoker was 118 ± 7.92 mmHg. This difference in BP was significant at P less than 0.05

Mean DBP of smoker was 87 ± 5.6 mmHg and non-smoker was 80 ± 7.5 mmHg. This difference in BP was significant at P value less than 0.05. Mean PP of smoker was 48.62 ± 7.2 mmHg and non-smoker was 37.7 ± 6.9 mmHg. This difference in BP was significant at P less than 0.05.

Various researchers have suggested the different effects of cigarette smoking on BP. Some Suggested that there is no association between smoking and BP. 29,30,31 Some suggested that BP of smokers was lower than the BP of non smokers 32,33,34 while others suggested that smoking increases BP. 35,36,37. In one study, the researchers in their study demonstrated the effect of cigarette smoking on BP They reported that there is increase in systolic as well as DBP along with heart rate (HR) after cigarette smoking whether tie cigarettes are of low-nicotine cigarettes or high-nicotine cigarettes.³⁵

Autonomic imbalance in smokers can be linked to the effect of nicotine – mediated stimulation of autonomic ganglia and adrenal medulla resulting in increased discharge in cardiac sympathetic fibers. This enhanced sympathetic activity increases HR, BP, and myocardial contractility by acting on β1-adrenergic receptor and also increases coronary vasomotor tone by acting on α2-adrenoceptor. 38,39,40,41 Some studies reported that PP is a better predictor of arterial wall stress due to BP than SBP and diastolic pressure. An increase in PP

creates more stress on the arterial wall and left ventricle. The increase stress leads to damage of the blood vessels and ventricular hypertrophy. Damage to blood vessels leads to more chance of the development of atherosclerosis, thrombosis, and heart failure.

^{42,43} This study reported that the mean PP of smokers was higher than non-smokers. Association between cigarette smoking and a wide PP may reflect either an increased peripheral resistance in smokers or the presence of aortic atherosclerosis and arterial inelasticity as smoking is a risk factor for atherosclerosis.⁴⁴

Smoking is a major risk factor for cardiovascular morbidity and mortality, and is considered to be the leading preventable cause of death in the world. Based on WHO estimates, tobacco continues to kill nearly 6 million people each year, including more than 600,000 passive smokers, through heart disease, lung cancer, and other illnesses; that is one and a half million more than the corresponding estimate for 1990. If current trends continue, the death toll is projected to reach more than the 8 million per year by 2030. 21 Smoking is associated with an increased risk of all types of cardiovascular disease, including coronary heart disease, ischemic stroke, peripheral artery disease and abdominal aortic aneurysm. In the present study, smoking was found to affect the resting and exercise HR responses in young male smokers. Smokers had elevated HR rest, a slower HR increase during exercise, impaired ability to reach their age-predicted HR max when compared to non smokers. Significant change between both groups was observed regarding resting heart rate in group A and B (77.7 ± 4.14 beats per minute (bpm) versus 71.4 ± 4.85 bpm) with P value 0.0001. Regarding heart rate after exercise in group A and B, it was (134.4 ± 6.5 versus 129.6 ± 7.4) with P value; 0.006, regarding to heart rate after 5 minute recovery from exercise in group A and B, it was (84 ± 7.7 versus 83 ± 7.4) with P value; 0.578.

Regarding time in exercise in group A and B, with P value, these results are in line with previously published data from young populations, 22 and they are also in agreement with many HR related studies of healthy middle aged populations, where smoking has been associated with increased resting HR values. 45,46 Smoking is associated with autonomic dysfunction and with selective alterations in cardiac autonomic control. More specifically, smoking, acting at peripheral sympathetic sites, increases circulating levels of catecholamine's, augments sympathetic outflow, and causes a long-term reduction in vagal drive. This sympathetic predominance, seen even in young heavy smokers, is also associated with impaired baroreflex functions, leading to a marked increase in HR rest.⁴⁷

Heart rate at sub maximal workload the HR value at a fixed sub-maximal aerobic workload (HR submax) is directly related with the increased metabolic demands imposed by the specific workload intensity. Thus, HRsubmax can be considered as an important maker of myocardial work, being inversely associated with exercise capacity, and cardiovascular health. 48 Our data indicated that there was a significant difference found between young smokers and non-smokers regarding their sub-maximal HR values, where smokers had significantly higher HR values.

There are few studies examining the effects of smoking on HR submax in healthy young adults. In some studies smoking was found to increase men's HR at a fixed sub maximal workload, 45 whereas elsewhere it was suggested that smokers have lower HR at sub maximal exercise, and others found no differences. Differences in methodology (e.g. definition of sub maximal workload, HR evaluation protocol, selection criteria for smokers, etc.) might have contributed to these divergent

findings. 49 In our study there was no difference between both groups regarding to recovery heart rate there are very few studies that have examined the association between smoking and HR rec in young adults. Kobayashi et al reported that young smokers had attenuated HR decline after sub-maximal exercise.⁵⁰

CONCLUSION

Smoking was found to affect young smokers' HR, increasing HR at rest, slowing HR in crease during exercise, and impairing their ability to reach the age predicted HRmax. In addition, smoking was associated with an attenuated HR decline during recovery. Smokers had a higher resting HR and showed a higher HR response during submaximal exercise compared to non smokers. Also, this study reported the effect of short duration of smoking with low pack year on BP and obtained the result of increased BP in smokers. This study can be helpful for making preventive strategies toward healthy life of young adults.

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Compliance with Ethical Standards.

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